

**WEST****The Contents of Case 09464303**

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Qnum	Query	DB Name	Thesaurus	Operator	Plural
Q1	(Stahl)[IN] OR (collard)[IN]	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q2	Q1 and complement	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q3	mbl or (mannose adj binding adj lectin)	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES
Q4	Q3 and (3f8 or 2a9 or hmb1.2 or hb-12621 or hb-12620 or hb-12619)	USPT,PGPB,JPAB,EPAB,DWPI	None	OR	YES

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conditions, LCL induced titers of IFN- $\gamma$  corresponding to >20,000 IFN- $\alpha$  units/mL medium, higher than established with other tested, established IFN- $\gamma$  inducers. Other desirable properties of this lectin, as discussed, also suggest that it will be of value for efficient large-scale IFN- $\gamma$  prodn.

=> end

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LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
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FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-4.12	-4.12

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NEWS 8 Mar 22 TRCTHERMO no longer available  
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=> s Stahl G7/au or collard C7/au  
L1 964 STAHL G7/AU OR COLLARD C7/AU

=> s l1 and complement  
L2 168 L1 AND COMPLEMENT

=> s l2 and ( (MBL) or (mannose (1N) binding (1N) lectin))  
L3 41 L2 AND ((MBL) OR (MANNOSE (1N) BINDING (1N) LECTIN))

=> dup rem l3  
PROCESSING COMPLETED FOR L3  
L4 21 DUP REM L3 (20 DUPLICATES REMOVED)

wo/2001/2212 B1

=> dis 14 1-21 ibib abs

L4 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2001:137043 CAPLUS  
DOCUMENT NUMBER: 134:188227  
TITLE: Inhibitors of the lectin complement pathway  
(LCP) and their use  
INVENTOR(S): Stahl, Gregory L.; Lekowski, Robert  
PATENT ASSIGNEE(S): The Brigham and Women's Hospital, Inc., USA  
SOURCE: PCT Int. Appl., 87 pp.  
CODEN: PIXKD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001012212	A1	20010222	WO 2000-US22123	20000814

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-148815P P 19990813

AB The invention relates to methods and products for regulating lectin complement pathway assocd. complement activation. The methods include both in vitro and in vivo methods for inhibiting lectin complement pathway assocd. complement activation. The methods are accomplished by contacting a mammalian cell having surface exposed MBL ligand with an effective amt. of a mannan binding lectin (MBL) receptor antagonist to inhibit lectin complement pathway assocd. complement activation. The mannan binding lectin receptor antagonist may be administered to a subject to prevent cellular injury mediated by lectin complement pathway assocd. complement activation. The products of the invention include compns. of a mannan binding lectin receptor antagonist. The mannan binding lectin receptor antagonist is an isolated mannan binding lectin that selectively binds to a human mannan binding lectin epitope and that inhibits lectin complement pathway assocd. complement activation.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 21 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 2001259476 MEDLINE  
DOCUMENT NUMBER: 21136395 PubMed ID: 11238665  
TITLE: A keratin peptide inhibits mannose-binding lectin.  
AUTHOR: Montalto M C; Collard C D; Buras J A; Reenstra W  
R; McClaine R; Gies D R; Rother R P; Stahl G L  
CORPORATE SOURCE: Department of Anesthesiology, Perioperative and Pain Medicine, Center for Experimental Therapeutics and Reperfusion Injury, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.  
CONTRACT NUMBER: F32 HL-103870 (NHLBI)  
HL-03854 (NHLBI)  
HL-56086 (NHLBI)  
SOURCE: JOURNAL OF IMMUNOLOGY, (2001 Mar 15) 166 (6) 4148-53.  
Journal code: IFB; 2985117R. ISSN: 0022-1767.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200105  
ENTRY DATE: Entered STN: 20010521  
Last Updated on STN: 20010521  
Entered Medline: 20010517

AB Complement plays a significant role in mediating endothelial injury following oxidative stress. We have previously demonstrated that the lectin complement pathway (LCP), which is initiated by deposition of the mannose-binding lectin (MBL), is largely responsible for activating complement on endothelial cells following periods of oxidative stress. Identifying functional inhibitors that block MBL binding will be useful in characterizing the role of the LCP in disease models. The human cytokeratin peptide SPGSGFGGGY has been identified as a molecular mimic of N-acetyl-D-glucosamine (GlcNAc), a known ligand of MBL. Thus, we hypothesized that this peptide would specifically bind to MBL and functionally inhibit the LCP on endothelial cells following oxidative stress. Using a BiAcure 3000 optical biosensor, competition experiments were performed to demonstrate that the peptide SPGSGFGGGY inhibits binding of purified recombinant human MBL to GlcNAc in a concentration-dependent manner. Solution affinity data generated by BiAcure indicate this peptide binds to MBL with an affinity (K(D)) of  $5 \times 10^{-5}$  mol/L. Pretreatment of human serum (30%) with the GlcNAc-mimicking peptide (10-50 microg/ml) significantly attenuated MBL and C3 deposition on human endothelial cells subjected to oxidative stress in a dose-dependent manner, as demonstrated by cell surface ELISA and confocal microscopy. Additionally, this decapeptide sequence attenuated complement-dependent VCAM-1 expression following oxidative stress. These data indicate that a short peptide sequence that mimics GlcNAc can specifically bind to MBL and functionally inhibit the proinflammatory action of the LCP on oxidatively stressed endothelial cells.

L4 ANSWER 3 OF 21 MEDLINE DUPLICATE 2  
ACCESSION NUMBER: 2001481748 MEDLINE  
DOCUMENT NUMBER: 21400864 PubMed ID: 11509633  
TITLE: Human IgA activates the complement system via the mannan-binding lectin pathway.  
AUTHOR: Roos A; Bouwman L H; van Gijlswijk-Janssen D J; Faber-Krol M C; Stahl G L; Daha M R  
CORPORATE SOURCE: Department of Nephrology, Leiden University Medical Center, Leiden, The Netherlands.. A.Roos@LUMC.NL  
SOURCE: JOURNAL OF IMMUNOLOGY, (2001 Sep 1) 167 (5) 2861-8.  
Journal code: 2985117R. ISSN: 0022-1767.  
PUB. COUNTRY: United States  
LANGUAGE: English; Article; (JOURNAL ARTICLE)

LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200112  
ENTRY DATE: Entered STN: 20010830  
Last Updated on STN: 20020122  
Entered Medline: 20011205

AB The recently identified lectin pathway of the complement system, initiated by binding of mannan-binding lectin (MBL) to its ligands, is a key component of innate immunity. MBL-deficient individuals show an increased susceptibility for infections, especially of the mucosal system. We examined whether IgA, an important mediator of mucosal immunity, activates the complement system via the lectin pathway. Our results indicate a dose-dependent binding of MBL to polymeric, but not monomeric IgA coated in microtiter plates. This interaction involves the carbohydrate recognition domain of MBL, because it was calcium dependent and inhibited by mannose and by mAb against this domain of MBL. Binding of MBL to IgA induces complement activation, as demonstrated by a dose-dependent deposition of C4 and C3 upon addition of a complement source. The MBL concentrations required for IgA-induced C4 and C3 activation are well below the normal MBL plasma concentrations. In line with these experiments, serum from individuals having mutations in the MBL gene showed significantly less activation of C4 by IgA and mannan than serum from wild-type individuals. We conclude that MBL binding to IgA results in complement activation, which is proposed to lead to a synergistic action of MBL and IgA in antimicrobial defense. Furthermore, our results may explain glomerular complement deposition in IgA nephropathy.

L4 ANSWER 4 OF 21 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 2001512912 MEDLINE  
DOCUMENT NUMBER: 21444721 PubMed ID: 11560858  
TITLE: Inhibition of mannose-binding  
lectin reduces postischemic myocardial reperfusion  
injury.  
AUTHOR: Jordan J E; Montalto M C; Stahl G L  
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion  
Injury, Department of Anesthesiology, Perioperative and  
Pain Medicine, Brigham and Women's Hospital, Harvard  
Medical School, Boston, MA 02115, USA.  
CONTRACT NUMBER: HL-10346 (NHLBI)  
HL-10387 (NHLBI)  
HL-52886 (NHLBI)  
HL-56086 (NHLBI)  
SOURCE: CIRCULATION, (2001 Sep 18) 104 (12) 1413-8.  
Journal code: DAW; 0147763. ISSN: 1524-4539.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200110  
ENTRY DATE: Entered STN: 20010919  
Last Updated on STN: 20011008  
Entered Medline: 20011004

AB BACKGROUND: Complement consists of a complex cascade of proteins involved in innate and adaptive immunity. The cascade can be activated through 3 distinct mechanisms, designated the classical, alternative, and lectin pathways. Although complement is widely accepted as participating in the pathophysiology of ischemia-reperfusion injury, the specific role of the lectin pathway has not been addressed. METHODS AND RESULTS: Monoclonal antibodies (mAbs; P7E4 and 14C3.74, IgG1kappa isotypes) were raised against rat mannose-binding lectin (MBL). Both mAbs recognized rMBL-A by Western analysis or surface plasmon resonance. P7E4, but not 14C3.74, exhibited a concentration-dependent inhibition of the lectin pathway, with maximal effect at 10  $\mu$ g/mL. In vivo, rats were subjected to 30 minutes of left coronary artery occlusion and 4 hours of reperfusion. Complement C3 deposition was greatly attenuated in hearts pretreated with P7E4 compared with 14C3.74-treated hearts. Pretreatment with P7E4 (1 mg/kg) significantly reduced myocardial creatine kinase loss (48%), infarct size (39%), and neutrophil infiltration (47%) compared with 14C3.74-treated animals. In addition, P7E4 pretreatment significantly attenuated the expression of proinflammatory genes (intercellular adhesion molecule-1, vascular cell adhesion molecule-1, and interleukin-6) after ischemia-reperfusion. CONCLUSIONS: The lectin complement pathway is activated after myocardial ischemia-reperfusion and leads to tissue injury. Blockade of the lectin pathway with inhibitory mAbs protects the heart from ischemia-reperfusion by reducing neutrophil infiltration and attenuating proinflammatory gene expression.

L4 ANSWER 5 OF 21 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 2001499911 MEDLINE  
DOCUMENT NUMBER: 21433399 PubMed ID: 11549596  
TITLE: Endothelial oxidative stress activates the lectin  
complement pathway: role of cytokeratin 1.  
AUTHOR: Collard C D; Montalto M C; Reenstra W R; Buras J  
A; Stahl G L  
CORPORATE SOURCE: Department of Anesthesiology, Perioperative, and Pain  
Medicine, Center for Experimental Therapeutics and  
Reperfusion Injury, Brigham and Women's Hospital, Harvard  
Medical School, Boston, MA 02115, USA.  
CONTRACT NUMBER: P32-HL103870 (NHLBI)  
HL-03854 (NHLBI)  
HL-52886 (NHLBI)  
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2001 Sep) 159 (3) 1045-54.  
Journal code: 3RS; 0370502. ISSN: 0002-9440.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200110  
ENTRY DATE: Entered STN: 20010911  
Last Updated on STN: 20011015  
Entered Medline: 20011011

AB Oxidative stress increases endothelial mannose-binding lectin (MBL) binding and activates the lectin complement pathway (LCP). However, the molecular mechanism of MBL binding to the endothelium after oxidative stress is unknown. Intermediate filaments have been previously reported to activate the classical complement pathway in an antibody-independent manner. We investigated whether oxidative stress increases human umbilical vein endothelial cell (HUVEC) cytokeratin 1 (CK1) expression and activates the

LCP via MBL binding to CK1. Reoxygenation (4 hours, 21% O<sub>2</sub>) of hypoxic HUVECs (24 hours, 1% O<sub>2</sub>) significantly increased CK1 mRNA (in situ hybridization) and membrane protein expression [enzyme-linked immunosorbent assay (ELISA)/confocal microscopy]. Incubating human serum (HS) with N-acetyl-D-glucosamine or anti-human MBL monoclonal antibody attenuated MBL and C3 deposition on purified CK1 (ELISA). CK1 and MBL were co-immunoprecipitated from hypoxic HUVECs reoxygenated in HS. Treatment with anti-human cytokeratin Fab fragments attenuated endothelial MBL and C3 deposition after oxidative stress (ELISA/confocal microscopy). We conclude that: 1) endothelial oxidative stress increases CK1 expression, MBL binding, and C3 deposition; 2) inhibition of MBL attenuates purified CK1-induced complement activation; and 3) anti-human cytokeratin Fab fragments attenuate endothelial MBL and C3 deposition after oxidative stress. These results suggest that MBL binding to endothelial cytokeratins may mediate LCP activation after oxidative stress.

L4 ANSWER 6 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:267818 BIOSIS  
DOCUMENT NUMBER: PREV200100267818  
TITLE: Epitope mapping monoclonal antibodies against human mannose binding lectin.  
AUTHOR(S): Zhao, Hui (1); Stahl, Gregory L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA, 02115 USA  
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A685.  
print.  
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001  
ISSN: 0892-6638.  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB MBL plays an important role in complement activation following endothelial oxidative stress. We have generated a panel of monoclonal antibodies (3F8, hMBL1.2 and 2A9) against human MBL. These antibodies are functional inhibitors, which can attenuate MBL-dependent C3 deposition after endothelial oxidative stress. The affinity of Fab fragments and whole IgG antibodies to MBL were very similar. However, Fab fragments of hMBL1.2 or 2A9 did not inhibit C3 deposition in a MBL dependent assay. Further, F(ab)<sub>2</sub> fragments of 2A9 or hMBL1.2 were functionally much less effective compared to whole IgG, suggesting that steric hindrance of these two antibodies are important for their inhibition of MBL binding. Fab and F(ab)<sub>2</sub> fragments of 3F8, on the other hand, were functionally as effective as the whole IgG. All three functional antibodies (3F8, hMBL1.2 and 2A9) bind to the carbohydrate recognition domain (CRD) of MBL based on protein sequencing and Western analysis of proteolytic fragments of MBL. MBL constructs consisting of sequential deletion of N- or C-terminal amino acids of the CRD region showed that the antibodies recognized different epitopes. Two disulfide bonds within the MBL monomer. Cys155 to Cys244 and Cys222 to Cys236 aid in stabilization of the CRD. Single, double and triple mutations of these cysteines showed that the disulfide bonds played a role in forming discontinuous epitopes for 3F8 and hMBL1.2. Epitope maps of these antibodies were further confirmed by biopanning using the PliTrx random peptide display library. Generating monoclonal antibodies against MBL will aid in the structure/function analysis of MBL and its role in inflammatory diseases.

L4 ANSWER 7 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:257627 BIOSIS  
DOCUMENT NUMBER: PREV200100257627  
TITLE: Regulation of pro-inflammatory genes by the lectin complement pathway following myocardial ischemia-reperfusion.  
AUTHOR(S): Jordan, James E. (1); Montalto, Michael C. (1); Lopes da Rosa, Jessica R. (1); Stahl, Gregory L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis St., Boston, MA, 02115 USA  
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A463.  
print.  
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001  
ISSN: 0892-6638.  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB Reperfusion of ischemic myocardium initiates an inflammatory-like process leading to additional tissue injury. Following myocardial ischemia and reperfusion (MI/R), complement is activated with a subsequent increase in pro-inflammatory cytokines and adhesion molecule gene expression. Increasing evidence suggests that the lectin complement pathway (LCP) is involved in initiating the complement cascade following MI/R. In this study, we assessed the result of inhibiting complement activation via the LCP on the expression of inflammatory genes (cytokines and adhesion molecules) following MI/R. Male Sprague-Dawley rats were subjected to 30 minutes of ischemia followed by 4 hours of reperfusion. Prior to occlusion, rats were treated with 1 mg/kg P7E4 (a mAb against rat mannose binding lectin-MBL A) or GS-1 (an isotype control antibody). Following 4 hours of reperfusion, the hearts were removed, washed in saline and the area at risk was frozen in liquid nitrogen. Total RNA was isolated using the acid-guanidinium thiocyanate extraction procedure and subjected to DNase treatment. Semi-quantitative RT-PCR was performed to assess mRNA levels of ICAM-1, TNF-alpha, iNOS, eNOS, SOD (Cu/Zn and Mn) GM-CSF and IL-1 alpha, in sham operated animals and those treated with GS-1 or P7E4 as described above. Ischemia-reperfusion resulted in the increased expression of ICAM-1\*, iNOS\*, TNF-alpha\*, GM-CSF\* and IL-1 alpha\* compared to sham operated controls. Inhibition of MBL A attenuated the increased expression for ICAM-1\*, GM-CSF\*, IL-1 alpha\* and iNOS. These data suggest that inhibition of MBL A and the lectin complement pathway results in the attenuation of inflammatory gene expression and that this gene regulation may be partially responsible for the protection from MI/R injury, \* p < 0.05.

L4 ANSWER 8 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:196301 BIOSIS  
DOCUMENT NUMBER: PREV200100196301

TITLE: Inhibition of mannose binding lectin reduces myocardial reperfusion injury: A role for the lectin complement pathway in cardiovascular disease.

AUTHOR(S): Jordan, James E. (1); Stahl, Gregory L. (1)

CORPORATE SOURCE: (1) Dept. of Anesthesia, CET and RI, Brigham and Women's Hospital, Boston, MA USA

SOURCE: Journal of the American College of Cardiology, (February, 2001) Vol. 37, No. 2 Supplement A, pp. 378A. print.

Meeting Info.: 50th Annual Scientific Session of the American College of Cardiology Orlando, Florida, USA March 18-21, 2001

ISSN: 0735-1097.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

L4 ANSWER 9 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:276725 BIOSIS

DOCUMENT NUMBER: PREV200100276725

TITLE: A peptide mimic of N-acetyl-D-glucosamine inhibits the lectin complement pathway following endothelial oxidative stress.

AUTHOR(S): Montalto, Michael C. (1); Collard, Charles D. (1); Buras, Jon A.; Reenstra, Wende R.; Geis, David; Rother, Russell P.; Stahl, Gregory L. (1)

CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis Street, Boston, MA, 02115 USA

SOURCE: PASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A339. print.

Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001

ISSN: 0892-6638.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Complement plays a significant role in mediating endothelial damage following oxidative stress. We have previously demonstrated that the lectin complement pathway (LCP), which is initiated by mannose binding lectin (MBL) deposition, is largely responsible for activating complement after endothelial oxidative stress. Identifying functional inhibitors of MBL will be useful in characterizing the role of the LCP following periods of oxidative stress. To date, peptide analogues specific for MBL have not been identified. The human cytokeratin peptide, SFGSGPGGGY, has previously been identified as a molecular mimic of N-acetyl-D-glucosamine (GlcNAc), a natural ligand of MBL. Thus, we hypothesized that the sequence SFGSGPGGGY would specifically bind MBL and functionally inhibit the LCP. Using a BIAcore 3000 optical biosensor, we performed competition experiments to demonstrate that the peptide SFGSGPGGGY can inhibit binding of recombinant human MBL to GlcNAc in a concentration dependent manner. Solution affinity data generated by BIAcore indicate this peptide binds to MBL with an affinity (KD) of 5 X 10-5 M. Pretreatment of human serum (30%) with the GlcNAc-mimicking peptide (10 - 50 mug/ml) significantly attenuated C3 deposition on oxidatively stressed endothelial cells in a dose dependent manner, as demonstrated by ELISA. Confocal microscopy experiments revealed that complement inhibition coincided with a decrease in MBL deposition. Additionally, this peptide significantly attenuated the complement-dependent expression of vascular cell adhesion molecule (VCAM)-1 as shown by ELISA. These data indicate that a short peptide sequence that mimics GlcNAc can specifically bind to MBL, with a physiologically relevant affinity, and functionally inhibit the pro-inflammatory action of the LCP on oxidatively stressed endothelial cells. Further, this is the first report to demonstrate that MBL is capable of specifically binding a non-carbohydrate ligand.

L4 ANSWER 10 OF 21 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 2001209693 MEDLINE

DOCUMENT NUMBER: 21195380 PubMed ID: 11298833

TITLE: Isolation, cloning and functional characterization of porcine mannose-binding lectin

AUTHOR: Agah A; Montalto M C; Young K; Stahl G L

CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham & Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.

CONTRACT NUMBER: HL52886 (NHLBI)

SOURCE: IMMUNOLOGY, (2001 Mar) 102 (3) 338-43.

Journal code: GH7; 0374672. ISSN: 0019-2805.

PUB. COUNTRY: England; United Kingdom

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200105

ENTRY DATE: Entered STN: 20010517

Last Updated on STN: 20010517

Entered Medline: 20010510

AB Binding of mannose-binding lectin (MBL), a C-type lectin, and its associated serine proteases, MASP-1 and MASP-2, to cell surface carbohydrates activates the lectin complement pathway. As MBL plays an important role in innate immunity, it has been cloned and characterized in several species. While the pig may be used as a source of organs/tissues for xenotransplantation, little is known about its MBL, thus, we report the isolation of three monomeric forms of MBL from porcine serum. Sodium dodecyl sulphate-polyacrylamide gel electrophoresis and Coomassie staining of reduced porcine MBL revealed the presence of three monomeric forms with approximate molecular masses of 30 000, 32 000 and 34 000. Protein sequencing identified these monomeric forms as one single protein, suggesting post-translational modification. Western blot analysis demonstrated the cross-reactivity of anti-human MBL polyclonal antibody with porcine MBL. A full-length porcine liver MBL cDNA was isolated and the predicted amino acid sequence exhibited 64.9% identity with human MBL and 50.2% and 36.7% identity with rat A and C MBL, respectively. Furthermore, Northern blot analysis demonstrated the presence of a single (approximately 1.4-1.6 kilobase pair) transcript in porcine liver. Addition of purified porcine MBL to MBL-deficient human sera augmented N-acetylglucosamine inhibitable C3 deposition to mannan-coated plates in a dose-dependent manner. Taken together, these data demonstrate

that porcine and human MBL are highly conserved, sharing structural and functional characteristics.

L4 ANSWER 11 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2001:276723 BIOSIS  
DOCUMENT NUMBER: PREV200100276723  
TITLE: Isolation and characterization of anti-rat mannose binding lectin antibodies.  
AUTHOR(S): Jordan, James E. (1); Morrissey, Margaret A. (1); Stahl, Gregory L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, 75 Francis St., Boston, MA, 02115 USA  
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A338. print.  
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001 ISSN: 0892-6638.  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

AB Complement is a major participant in post-ischemic reperfusion injury. The classical and alternative complement pathways have been extensively studied; however, the role of the lectin complement pathway (LCP) in inflammation has not been investigated since inhibitors of MBL have not been described. In order to delineate the participation of the LCP, we generated antibodies to rat mannose binding lectin (MBL). In this study, antibodies (Ab) were produced and characterized for potential use in further dissecting the role of the LCP in rat models of human disease. Polyclonal and monoclonal antibodies were produced in rabbits and mice using purified MBL isolated from rat sera by affinity chromatography and standard immunization techniques. All antibodies were screened using 2 different ELISA systems (binding to mannan or BSA coupled to N-acetyl-D-glucosamine; BSA-GlcNAc) to detect complement activation and deposition of C3. Monoclonal Ab P7E4 inhibited C3 deposition onto BSA-GlcNAc coated plates in a concentration-dependent manner with maximal inhibition (approx 80%) occurring at 10 µg/mL. Similarly, Fab fragments of the polyclonal antibody inhibited complement deposition in a concentration dependent manner. Western blot analysis was performed to determine the isoform(s) of rat MBL that the antibodies recognized. While the polyclonal Ab recognized multiple oligomers of A and C isoforms under non-reducing conditions, mAb P7E4 only recognized the A isoform. Similar results were obtained under reducing conditions in that the polyclonal Ab recognized both forms while only the A isoform was recognized by P7E4. These data confirm that the antibodies recognize rat mannose binding lectin, inhibit the function of MBL and may serve as useful reagents in studying the lectin complement pathway in rat models of human disease.

L4 ANSWER 12 OF 21 MEDLINE MEDLINE DUPLICATE 6  
ACCESSION NUMBER: 2001422305 MEDLINE  
DOCUMENT NUMBER: 21167477 PubMed ID: 11266613  
TITLE: Ulex europaeus agglutinin II (UEA-II) is a novel, potent inhibitor of complement activation.  
AUTHOR: Lekowski R; Collard C D; Reenstra W R; Stahl G L  
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.  
CONTRACT NUMBER: GM-07592 (NIGMS)  
HL-03854 (NHLBI)  
HL-52886 (NHLBI)  
HL-56086 (NHLBI)  
SOURCE: PROTEIN SCIENCE, (2001 Feb) 10 (2) 277-84.  
Journal code: BNW; 9211750. ISSN: 0961-8368.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200107  
ENTRY DATE: Entered STN: 20010730  
Last Updated on STN: 20010730  
Entered Medline: 20010726

AB Complement is an important mediator of vascular injury following oxidative stress. We recently demonstrated that complement activation following endothelial oxidative stress is mediated by mannose-binding lectin (MBL) and activation of the lectin complement pathway. Here, we investigated whether nine plant lectins which have a binding profile similar to that of MBL competitively inhibit MBL deposition and subsequent complement activation following human umbilical vein endothelial cell (HUVEC) oxidative stress. HUVEC oxidative stress (1% O<sub>2</sub>, 24 hr) significantly increased Ulex europaeus agglutinin II (UEA-II) binding by 72 ± 9% compared to normoxic cells. UEA-II inhibited MBL binding to HUVEC in a concentration-dependent manner following oxidative stress. Further, MBL inhibited UEA-II binding to HUVEC in a concentration-dependent manner following oxidative stress, suggesting a common ligand. UEA-II (< or = 100 µmol/L) did not attenuate the hemolytic activity, nor did it inhibit C3a des Arg formation from alternative or classical complement pathway-specific hemolytic assays. C3 deposition (measured by ELISA) following HUVEC oxidative stress was inhibited by UEA-II in a concentration-dependent manner (IC<sub>50</sub> = 10 µmol/L). UEA-II inhibited C3 and MBL co-localization (confocal microscopy) in a concentration-dependent manner on HUVEC following oxidative stress (IC<sub>50</sub> approximately 1 µmol/L). Finally, UEA-II significantly inhibited complement-dependent neutrophil chemotaxis, but failed to inhibit fMLP-mediated chemotaxis, following endothelial oxidative stress. These data demonstrate that UEA-II is a novel, potent inhibitor of human MBL deposition and complement activation following human endothelial oxidative stress.

L4 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2000:420986 CAPLUS  
DOCUMENT NUMBER: 133:57580  
TITLE: Methods and products for regulating lectin complement pathway associated complement activation  
INVENTOR(S): Stahl, Gregory L.; Collard, Charles D.

PATENT ASSIGNEE(S): Brigham and Women's Hospital, Inc., USA  
SOURCE: PCT Int. Appl., 68 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000035483	A1	20000622	WO 1999-US29919	19991215
W: CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1140171	A1	20011010	EP 1999-967362	19991215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-112390P P 19981215  
WO 1999-US29919 W 19991215

AB The invention relates to methods and products for regulating lectin complement pathway assocd. complement activation. The methods include both in vitro and in vivo methods for inhibiting lectin complement pathway assocd. complement activation. The methods are accomplished by contacting a mammalian cell having surface exposed MBL ligand with an effective amt. of a mannan binding lectin inhibitor to inhibit lectin complement pathway assocd. complement activation. The mannan binding lectin inhibitor may be administered to a subject to prevent cellular injury mediated by lectin complement pathway assocd. complement activation. The products of the invention include compns. of a mannan binding lectin inhibitor. The mannan binding lectin inhibitor is an isolated mannan binding lectin binding peptide that selectively binds to a human mannan binding lectin epitope and that inhibits lectin complement pathway assocd. complement activation. The products also include hybridoma cell lines and pharmaceutical compns.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 21 MEDLINE DUPLICATE 7  
ACCESSION NUMBER: 2000255148 MEDLINE  
DOCUMENT NUMBER: 20255148 PubMed ID: 10793066  
TITLE: Complement activation after oxidative stress: role of the lectin complement pathway.  
AUTHOR: Collard C D; Vakeva A; Morrissey M A; Agah A; Rollins S A; Reenstra W R; Buras J A; Meri S; Stahl G L  
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Boston, Massachusetts 02115, USA.  
CONTRACT NUMBER: HL-03854 (NHLBI)  
HL-52886 (NHLBI)  
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2000 May) 156 (5) 1549-56. Journal code: 3RS; 0370502. ISSN: 0002-9440.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 200006  
ENTRY DATE: Entered STN: 20000616  
Last Updated on STN: 20000616  
Entered Medline: 20000602

AB The complement system plays an important role in mediating tissue injury after oxidative stress. The role of mannose-binding lectin (MBL) and the lectin complement pathway (LCP) in mediating complement activation after endothelial oxidative stress was investigated. iC3b deposition on hypoxic (24 hours; 1% O(2))/reoxygenated (3 hours; 21% O(2)) human endothelial cells was attenuated by N-acetyl-D-glucosamine or D-mannose, but not L-mannose, in a dose-dependent manner. Endothelial iC3b deposition after oxidative stress was also attenuated in MBL-deficient serum. Novel, functionally inhibitory, anti-human MBL monoclonal antibodies attenuated MBL-dependent C3 deposition on mannan-coated plates in a dose-dependent manner. Treatment of human serum with anti-MBL monoclonal antibodies inhibited MBL and C3 deposition after endothelial oxidative stress. Consistent with our in vitro findings, C3 and MBL immunostaining throughout the ischemic area at risk increased during rat myocardial reperfusion in vivo. These data suggest that the LCP mediates complement activation after tissue oxidative stress. Inhibition of MBL may represent a novel therapeutic strategy for ischemia/reperfusion injury and other complement-mediated disease states.

L4 ANSWER 15 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2000:389415 BIOSIS  
DOCUMENT NUMBER: PREV200000389415  
TITLE: Endothelial oxidative stress increases cytokeratin 1 (K1) expression and human mannose-binding lectin (MBL) deposition.  
AUTHOR(S): Collard, C. D. (1); Montalto, M. (1); Stahl, G. L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, CET and RI, Harvard Medical School, Boston, MA USA  
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp. 85. print.  
Meeting Info.: XVIIIth International Complement Workshop Salt Lake City, Utah, USA July 23-27, 2000  
ISSN: 0162-3109.  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

L4 ANSWER 16 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2000:389411 BIOSIS  
DOCUMENT NUMBER: PREV200000389411  
TITLE: Characterization of monoclonal antibodies (mAb) against native and recombinant human mannose-binding lectin (MBL).  
AUTHOR(S): Zhao, H. (1); Stahl, G. L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, Boston, MA USA  
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp. 83. print.



Meeting Info.: XVIIIth International Complement Workshop  
Salt Lake City, Utah, USA July 23-27, 2000  
ISSN: 0162-3109.

DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

L4 ANSWER 17 OF 21 MEDLINE DUPLICATE 8

ACCESSION NUMBER: 2000162174 MEDLINE  
DOCUMENT NUMBER: 20162174 PubMed ID: 10698348  
TITLE: Complement activation following oxidative stress.  
AUTHOR: Collard C D; Lekowski R; Jordan J E; Agah A; Stahl G L  
CORPORATE SOURCE: Center for Experimental Therapeutics and Reperfusion Injury, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.  
CONTRACT NUMBER: GM-07592 (NIGMS)  
HL-03854 (NHLBI)  
HL-52886 (NHLBI)  
SOURCE: MOLECULAR IMMUNOLOGY, (1999 Sep-Oct) 36 (13-14) 941-8.  
Ref: 70  
PUB. COUNTRY: Journal code: NG1; 7905289. ISSN: 0161-5890.  
ENGLAND: United Kingdom  
Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200003  
ENTRY DATE: Entered STN: 20000327  
Last Updated on STN: 20000327  
Entered Medline: 20000316

AB It is clear that complement plays an important role in the inflammatory process following oxidative stress in cellular and animal models. Clinical trials underway with novel complement inhibitors will establish the potential therapeutic benefit of complement inhibition in human disease. For as much as we understand about the role of complement in disease states, many questions remain. How is complement activated on endothelial cells following oxidative stress? What is the ligand for MBL on endothelial cells following oxidative stress? Will inhibition of MBL provide tissue protection to the extent observed with other complement inhibitors such as sC1r or anti-C5 mAbs? These questions and more will undoubtedly be answered in the next millennium.

L4 ANSWER 18 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1999:384292 BIOSIS  
DOCUMENT NUMBER: PREV199900384292  
TITLE: Mannose-binding lectin  
co-localizes with complement in atherosclerotic human coronary arteries: A novel role for the lectin complement pathway in human cardiovascular disease.  
AUTHOR(S): Vakeva, A. (1); Collard, C. D.; Laine, P.; Morse, D. S.; Paavonen, T.; Meri, S. (1); Kovanen, P.; Stahl, G. L.  
CORPORATE SOURCE: (1) Haartman Institute, Department of Bacteriology and Immunology, University of Helsinki, Helsinki Finland  
SOURCE: Molecular Immunology, (March-April, 1999) Vol. 36, No. 4-5, pp. 302.  
Meeting Info.: 7th European Meeting on Complement in Human Disease Helsinki, Finland June 17-20, 1999  
ISSN: 0161-5890.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L4 ANSWER 19 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1999:167550 BIOSIS  
DOCUMENT NUMBER: PREV199900167550  
TITLE: Purification, characterization and cDNA sequencing of porcine mannose-binding lectin (MBL).  
AUTHOR(S): Agah, A.; Young, K.; Stahl, G. L.  
CORPORATE SOURCE: CET and RI, Dep. Anesthesia, Brigham and Women's Hosp., Harvard Med. Sch., Boston, MA 02115 USA  
SOURCE: FASEB Journal, (March 12, 1999) Vol. 13, No. 4 PART 1, pp. A284.  
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99 Washington, D.C., USA April 17-21, 1999  
ISSN: 0892-6638.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L4 ANSWER 20 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1999:395748 BIOSIS  
DOCUMENT NUMBER: PREV199900395748  
TITLE: Endothelial reoxygenation activates the lectin complement pathway: Inhibition with anti-human mannose binding lectin (MBL) therapy.  
AUTHOR(S): Collard, C. D. (1); Agah, A. (1); Buras, J. A. (1); Reenstra, W. R. (1); Morrissey, M. M. (1); Stahl, G. L. (1)  
CORPORATE SOURCE: (1) Center for Experimental Therapeutics and Reperfusion Injury, Dept. of Anesthesiology, Pain and Perioperative Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA USA  
SOURCE: Molecular Immunology, (March April, 1999) Vol. 36, No. 4-5, pp. 278.  
Meeting Info.: 7th European Meeting on Complement in Human Disease Helsinki, Finland June 17-20, 1999  
ISSN: 0161-5890.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L4 ANSWER 21 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2000:24535 BIOSIS  
DOCUMENT NUMBER: PREV200000024535  
TITLE: Ulex europaeus agglutinin II (UEA-II) is a novel, potent inhibitor of complement activation on human endothelial cells.

AUTHOR(S): Lekowski, Robert W. (1); R ra, Wende R.; Collard, Charles D.; Stahl, Gregory L.  
 CORPORATE SOURCE: (1) Brigham and Women's Hosp, Harvard Med Sch, Boston, MA USA  
 SOURCE: Circulation, (Nov. 2, 1999) Vol. 110, No. 18 SUPPL., pp. 1.259.  
 Meeting Info.: 72nd Scientific Sessions of the American Heart Association Atlanta, Georgia, USA November 7-10, 1999  
 ISSN: 0009-7322.  
 DOCUMENT TYPE: Conference  
 LANGUAGE: English

=> s (3f8 or 2a9 or hmb11.2 or HB-12621 or HB-12620 or hb-12619) and ( (MBL) or (mannose (1N) binding (1N) lectin))  
 UNMATCHED LEFT PARENTHESIS '(3F8'  
 The number of right parentheses in a query must be equal to the number of left parentheses.

=> s (3f8 or 2a9 or hmb11.2 or HB-12621 or HB-12620 or hb-12619) and ( (MBL) or (mannose (1N) binding (1N) lectin))  
 L5 3 (3F8 OR 2A9 OR HMBL1.2 OR HB-12621 OR HB-12620 OR HB-12619) AND ((MBL) OR (MANNOSE (1N) BINDING (1N) LECTIN))

=> dup rem l5  
 PROCESSING COMPLETED FOR L5  
 L6 3 DUP REM L5 (0 DUPLICATES REMOVED)

=> dis l6 1-3 ibib abs

L6 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
 ACCESSION NUMBER: 2001:267818 BIOSIS  
 DOCUMENT NUMBER: PREV200100267818  
 TITLE: Epitope mapping monoclonal antibodies against human mannose binding lectin.  
 AUTHOR(S): Zhao, Hui (1); Stahl, Gregory L. (1)  
 CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA, 02115 USA  
 SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A685.  
 print.  
 Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001  
 ISSN: 0892-6638.  
 DOCUMENT TYPE: Conference  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB MBL plays an important role in complement activation following endothelial oxidative stress. We have generated a panel of monoclonal antibodies (3F8, hMBL1.2 and 2A9) against human MBL. These antibodies are functional inhibitors, which can attenuate MBL-dependent C3 deposition after endothelial oxidative stress. The affinity of Fab fragments and whole IgG antibodies to MBL were very similar. However, Fab fragments of hMBL1.2 or 2A9 did not inhibit C3 deposition in a MBL dependent assay. Further, F(ab)2 fragments of 2A9 or hMBL1.2 were functionally much less effective compared to whole IgG, suggesting that steric hindrance of these two antibodies are important for their inhibition of MBL binding. Fab and F(ab)2 fragments of 3F8, on the other hand, were functionally as effective as the whole IgG. All three functional antibodies (3F8, hMBL1.2 and 2A9) bind to the carbohydrate recognition domain (CRD) of MBL based on protein sequencing and Western analysis of proteolytic fragments of MBL. MBL constructs consisting of sequential deletion of N- or C-terminal amino acids of the CRD region showed that the antibodies recognized different epitopes. Two disulfide bonds within the MBL monomer. Cys155 to Cys244 and Cys222 to Cys236 aid in stabilization of the CRD. Single, double and triple mutations of these cystidines showed that the disulfide bonds played a role in forming discontinuous epitopes for 3F8 and hMBL1.2. Epitope maps of these antibodies were further confirmed by biopanning using the PhiTrx random peptide display library. Generating monoclonal antibodies against MBL will aid in the structure/function analysis of MBL and its role in inflammatory diseases.

L6 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS  
 ACCESSION NUMBER: 2000:420986 CAPLUS  
 DOCUMENT NUMBER: 133:57580  
 TITLE: Methods and products for regulating lectin complement pathway associated complement activation  
 INVENTOR(S): Stahl, Gregory L.; Collard, Charles D.  
 PATENT ASSIGNEE(S): Brigham and Women's Hospital, Inc., USA  
 SOURCE: PCT Int. Appl., 68 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000035483	A1	20000622	WO 1999-US29919	19991215
W: CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1140171	A1	20011010	EP 1999-967362	19991215
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
PRIORITY APPLN. INFO.:			US 1998-112390P	P 19981215
			WO 1999-US29919	W 19991215

AB The invention relates to methods and products for regulating lectin complement pathway assocd. complement activation. The methods include both in vitro and in vivo methods for inhibiting lectin complement pathway assocd. complement activation. The methods are accomplished by contacting a mammalian cell having surface exposed MBL ligand with an effective amt. of a mannan binding lectin inhibitor to inhibit lectin complement pathway assocd. complement activation. The mannan binding lectin inhibitor may be administered to a subject to prevent cellular injury mediated by lectin complement pathway assocd. complement activation. The products of the invention include compns. of a mannan binding lectin inhibitor. The mannan binding lectin inhibitor is an isolated mannan binding lectin binding peptide that selectively binds to a human mannan binding lectin epitope and that inhibits lectin complement pathway assocd. complement activation. The products also include

hybridoma cell lines and pharmaceutical chemicals.  
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2000:389411 BIOSIS  
DOCUMENT NUMBER: PREV200000389411  
TITLE: Characterization of monoclonal antibodies (mAb) against  
native and recombinant human mannose-  
binding lectin (MBL).  
AUTHOR(S): Zhao, H. (1); Stahl, G. L. (1)  
CORPORATE SOURCE: (1) Brigham and Women's Hospital, Harvard Medical School,  
Boston, MA USA  
SOURCE: Immunopharmacology, (August, 2000) Vol. 49, No. 1-2, pp.  
83. print.  
Meeting Info.: XVIIIth International Complement Workshop  
Salt Lake City, Utah, USA July 23-27, 2000  
ISSN: 0162-3109.  
DOCUMENT TYPE: Conference  
LANGUAGE: English  
SUMMARY LANGUAGE: English

=> end  
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